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SEIZURE COMPLICATING INTERSCALENE BRACHAIL PLEXUS BLOCK

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ABSTRACT
We describe a case of seizure occurring immediately after completion of interscalene brachial plexus block, using 20mls mixture of 10mls of 0.5% bupivacaine and 10mls of 2% lidocaine with adrenaline for post operative analgesia. Seizure occurred despite negative test aspiration and non response to the use of 0.5mls of 2% lidocaine with adrenaline as intravascular markers, Seizures was aborted with the use of 10mg diazepam. We conclude that this seizure was due to inadvertent intravascular deposition of the mixture of local anaesthetic agent used despite the precautionary measures.

INTRODUCTION

Local anaesthetic systemic toxicity (LAST) ranges from mild systemic manifestation like auditory change, circum-oral numbness, metallic taste and agitation to central nervous system (CNS) effects such as seizures, coma, and respiratory arrest associated cardiovascular events include: hypertension, hypotension, tachycardia, bradycardia, ventricular arrhythmia and cardiac arrest [1].

The degree of systemic absorption depends on the dose of the local anaesthetic administered, site of block, vascularity of the injection site, use of adrenaline, patient morbid factors like cardiac, renal or hepatic dysfunction, as well as the physiochemical properties of the drug [2]. The close relationship between the nerves and blood vessels in the neurovascular bundle makes systemic manifestation possible either by direct deposition or by re-absorption. This is especially possible in the interscalene block site because of the nearness of the vertebral and subclavian arteries to the brachial plexus in compact the posterior triangle of the neck, which makes local anaesthesia systemic toxicity a possibility.

Test aspiration, although recommended sometimes fail to identify intravascular placement in up to 2% of patients [3]. We present a case report of seizure complicating interscalene brachial plexus block after the deposition of local anaesthetic solution despite negative aspiration of blood and the use of intravascular marker which are not 100% effective in preventing intravascular deposition. This is worthy of note to caution practitioners of regional anaesthesia.

CASE REPORT

A 28 year old female patient, who was involved in Road traffic accident and sustained fracture of the right humerus 6 months prior to presentation. She was admitted and scheduled for locked intramedullary nail, on account of deformity and pain on the right arm using Surgical Implant Generation Network (SIGN) nailing and to harvest bone graft from the tibia bone. There was no loss of consciousness, or bleeding from craniofacial orifices, no history of recent seizure.

In the pre –anaesthetic review, she was not a known hypertensive, diabetic, or seizure disorder patient. There was no past history of surgery or exposure to anaesthesia, and no known food

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or drug allergies. The patient neither smoked nor took alcohol.

Examination revealed a young lady who was calm; she was afebrile, not pale, anicteric, acyanosed and was not dehydrated. Cardiovascular examination revealed a pulse rate of 88/min and blood pressure of 120/80mmHg. Respiratory rate was 18cycles/min with vesicular breath sounds. The airway examination showed that mouth opening was adequate and there was good flexion/extension of the neck. Oropharnygeal examination was in keeping with Mallampatti 1, while American Society Anaesthesiologist Assessment of the physical health status was 1.

Her full blood count, electrolyte, urea and Creatinine and urinalysis were all within normal range. She was found positive to hepatitis B surface antigen (HbBAg). Patient was instructed to have an overnight fast. Grouping and cross matching of 2 units of blood was carried out. She was counselled for general anaesthesia with interscalene brachial plexus for post operative pain management.

Her baseline vital signs were (PR 102bpm, BP 139/68 mmHg, SpO_2 97-99% in room air). Intravenous access was secured with 18G cannular and normal saline infusion on the left hand. Anaesthetic machine check was done and items/drugs for resuscitation were also made available.

Using the modified Winnie's approach, a nerve stimulator by B-Braun HNS 12 with a 23G stimuplex needle, an evoked motor potential of the triceps muscle was observed at 1.5cm with a current of 1.5mA current. This was tailed down to 0.5mA and the evoked motor response was sustained. Test aspiration was done check needle placement, after which 0.5mls of 2% lidocaine with adrenaline was injected with no untoward increase in base line heart rate. A local anaesthetic mixture of a 20mls solution containing 10mls of 0.5% bupivacaine and 10mls of 2% lidocaine in adrenaline was deposited in 5mls aliquots after negative aspiration, with a positive RAJ response. There was no increased resistance during drug administration which indicative of intra-neural deposition. Within seconds of complete deposition of 20mls of the local anaesthetic as stimuplex needle was withdrawn, patient was noticed to be having generalized tonic-clonic seizures. Vital signs at the time of the seizure were (pulse rate 152/min, Blood pressure 121/53mmHg, $Sp0_2$ 94-97% in room air). Seizures were aborted with intravenous diazepam 10mg and 100% oxygen administered via facemask. It lasted 60 seconds, thereafter patient was conscious, well oriented in Time Place and Person and obeyed command, vital signs were (pulse rate132/min, Blood pressure 118/78mmHg, $Sp0_2$ of 99-100% in 100% of supplemental oxygen). The patient could not recall the seizure episode.

Surgery proceeded using general anaesthesia, she was pre-oxygenated with 100% oxygen and induction was achieved with 240mg of sodium-thiopentone. Laryngoscopy and endotracheal intubation were facilitated by 100mg of suxamethonium using size 7 mm cuffed endotracheal tubes. Anaesthesia was maintained with halothane 0.6-1%, atracurium 30mg using Intermittent Positive Pressure Ventilation (IPPV). Pain was managed with ketorolac 30mg, pethidine 50mg.

Intraoperative vital signs were pulse rate between154-126/min, blood pressure ranged between 88/53-126/95mmHg, and $Sp0_2$98-100%. At the end of surgery, residual neuromuscular block was antagonized and patient tracheal extubated with subsequent administration of 100% oxygen via facemask. Surgery lasted 2hours 15mins and was uneventful. First analgesic request was 3hour post operatively. First day post operative period was uneventful; patient was discharged home after 10days

DISCUSSION

Seizure in this case could be due to unintentional intermittent intravascular deposition of local anaesthetic agent during

interscalene block since onset time of seizure was about 2 minutes from the beginning of deposition of the local anaesthetic agent. Seizures occurring less than 60 seconds from the onset of deposition are due to accidental intravascular deposition particularly with injection into the carotid or vertebral arteries), premonitory symptoms can be bypassed and the patient can rapidly develop seizure. That occurring within 1-5 min may be due to either intermittent intravascular injection, excess systemic absorption or lower extremity intravascular injection with increased circulation time[4]. Seizure occurring at about 15 minutes or more may be due to either administration of local anaesthetic (LA) outside the maximum safe dose [4, 5] or sequel to slow metabolism. The seizures in this case could not have resulted because of local anaesthetic over dose, only 10mls of 0.5% plain bupivacaine (50mg) was administered; maximum safe dose for this adult should be 225mg (3mg/kg) [6]. While the dose of 2% lignocaine with adrenaline in 10mls is 20mg; maximum safe dose is 500mg (7mg/kg) [6]. There was no medical history of recurrent seizure or suffering from epilepsy.

Seizure occurred despite precautionary measures. Seizures are commonly associated with brachial plexus blockade, particularly with the interscalene and supraclavicular approaches, where local anaesthetics may be unintentionally injected into an artery supplying the brain. Incidence as high as 79 in 10,000 from a single institutional database has been documented [7].

Prevention of local anaesthetic systemic toxicity should be done by limiting the opportunity for intra- vascular injection or tissue uptake of local anaesthetic. This is best accomplished by early detection. Should an intravascular injection occur; it should ideally involve the lowest possible dose of local anaesthetic and consequently the least manifestation of LAST. There is no ideal method that prevents LAST. Aspirating the needle or catheter before each injection, may fail to identify intravascular placement in up to 2% of patients [3]. With regard to the use of intravascular markers, various options have been described but only fentanyl and adrenaline have met suggested standards of reliability and applicability by the American society of Regional Anaesthesia. Intravenous fentanyl produces sedation [8] while 10 to 15 µg/ml of adrenaline has a positive predictive value and 80% sensitivity in detecting intravascular injection in adults if heart rate increases by 10 beats per minute or higher or systolic blood pressure in- creases by 15 mm Hg or higher [9]. In children, intravascular adrenaline 0.5µg/kg is associated with at least a 15-mm Hg increase in systolic blood pressure. Nevertheless, adrenaline test doses are unreliable in the elderly, in patients who are sedated, those on ?-blockers, and in patients under general or neuraxial anaesthesia[1]. The marker of intravascular injection in this case, was the use of 0.5mls of lignocaine with adrenaline which was expected to increase the heart rate by$\geq$ 20% from the base line value. Incremental injection of 3 to 5mL of local anaesthetic with a pause for at least one circulation time before further injection is another alternative but this should be balanced against the risk of a prolonged duration of injection with the attendant risk of needle tip displacement. This could result in intravascular displacement or failed block.

The cardiovascular and central nervous systems are the major sites of toxicity. The latter is more sensitive to LA toxicity than the former [10], ie CNS intoxication usually manifests before signs of cardiovascular compromise except in cases of bupivacaine intoxication[11]. CNS intoxication is characterized by a two staged pathophysiologic process: shivering, muscle twitching and tremor precede tonic-clonic seizures. As plasma level of local anaesthetic agent increases it preferentially blocks inhibitory central pathways while leaving excitatory neurones unopposed thus resulting in seizures [12]. In the second stage, increasing local anaesthetic concentration, blocks both inhibitory and excitatory pathways leading to generalized CNS depression, the latter

results in hypoventilation and respiratory arrest. The index case manifested only the first stage CNS symptoms probably due to the fact that the serum concentration was not excessive. The pathophysiology of the cardiovascular system toxicity can also be considered in two stages. In the first stage during the CNS excitatory phase, activation of the sympathetic NS can lead to tachycardia and hypertension which may mask the direct local anaesthetic mediated myocardial effects. In the second stage as the blood level of local anaesthetic increases, bradycardia, asystole, decreased contractility; hypotension and cardiovascular collapse supersede the sympathetic mediated action. In this index case there was involvement of the first stage of the cardiovascular system with a heart rate of 152/minute (tachycardia) but no evidence of the second stage, which should have manifested with cardiovascular collapse. It should be pointed out that tachycardia alone without elevated blood pressure could have resulted from the pain of the procedure or indeed other sources of catecholamine release. There was therefore only a first stage involvement of the central nervous and cardiovascular systems.

Could the seizure have resulted because the method of nerve localization was by peripheral nerve stimulator which is a blind procedure? Ultrasound guidance may reduce the frequency of vascular puncture. However, there are no Randomized Control Trials (RCT) that con?rm or refute an actual reduction of LAST by this method [13]. Two large case series present con?icting results. One found a statistically signi?cant reduction in the number of vascular punctures occurring under Ultrasound Guided Regional Anaesthesia (UGRA) versus peripheral nerve stimulation, but no difference in LAST [14]. The other series reported a signi?cant reduction in seizures with ultrasound-assisted nerve localization compared to peripheral nerve stimulation [15], some case reports have described symptomatic intravascular injection despite its use [16]. Thus, the prevention of intravascular injection is perhaps best accomplished with a combination of UGRA and epinephrine test dosing. The practitioner's vigilance and prompt intervention is of utmost importance in the management of LAST.

The Cardiovascular (CV)/ Central Nervous System (CNS) ratio describes the dose required to produce cardiac arrhythmias versus that required to produce seizures. This ratio tends to be lower with bupivacaine compared with lidocaine, which implies a reduced safety margin for the potent local anaesthetic agents when detecting impending cardiac toxicity based on premonitory CNS signs. The more potent local anesthetics indeed generate arrhythmias at lower concentrations compared with lidocaine and mepivacaine. It is advised that agents like ropivacaine, levobupivacaine which are also long acting but with slightly higher CV/CNS ratio to bupivacaine should be used because of their better safety margin[17]. But there are also documented case reports of convulsion with the use of ropivacaine for interscalene blocks[18,19]

Treatment priorities for LAST include airway management, circulatory support, and measures aimed at reducing the systemic effects of local anesthetics. If seizures occur, they should be rapidly controlled to prevent injury to the patient and acidosis. Recent clinical data from patients experiencing local anaesthetic-induced convulsions demonstrated rapid development of hypoxia, hypercarbia, and acidosis with bupivacaine within a minute of the onset of convulsions[20]. These observations suggest that oxygen consumption and carbon dioxide production are greatly increased during local anaesthetic convulsions and emphasize the importance of immediate and effective ventilation with oxygen to avoid cardiac arrest. If not treated immediately, convulsions with simultaneous hypoxia, hypercarbia, and acidosis plus myocardial depression from the direct effects of the local anaesthetic may result in cardiac arrhythmias, bradycardia, asystole, ventricular fibrillation, or cardiac arrest. If

cardiac arrest occurs, successful outcome may require prolonged resuscitative efforts.

Diazepam was used here to abort the seizures because it is considered the ideal drug as it has limited potential for cardiac depression. However when benzodiazepines are used for anxiolysis, they may displace bupivacaine from protein binding sites. This sharp increase in the free plasma fraction may enhance the potential for CNS toxicity [21]. In the absence of any readily available benzodiazepine, propofol or thiopental are acceptable alternatives; however, their potential for worsening existing hypotension or cardiac depression requires using the lowest effective doses.

If tonic-clonic movements persist despite these measures, small doses of succinylcholine may be considered to rapidly stop muscular activity. This is because continued seizure activity exacerbates hypoxia and systemic acidosis. Seizure activity and acidosis will persist unless interrupted with a sedative hypnotic agent. Maintenance of cardiac output and oxygen delivery to tissues is critical for prevention and treatment of acidosis. Time to administer Intralipids is controversial; it is sometimes reserved for cases when advanced cardiac life support is unsuccessful[1].

The choice of interscalene brachial plexus block and general anaesthesia as the anaesthetic technique was influenced by the surgeon's decision to harvest bone graft from the tibia bone. This practice is a multimodal method of pain management which is a current concept of pain management, this result in better analgesic profile with reduced incidence of side effects. Without the tibia bone graft harvest, the interscalene brachial plexus block alone would have been adequate. General anaesthesia as an anaesthetic technique is not out of place but the choice of interscalene block and general anaesthesia was expected to provide prolonged post operative analgesia from the interscalene block which also reduced the doses of drugs used during general anaesthesia. Peripheral nerve block is normally performed before inducing general anaesthesia because the patient becomes a monitor of herself in order to detect complications associated with the procedure. The short post operative analgesia (3hours) resulted from the bone graft site pain while patient was still pain free from the operated upper limb till the 8th hour post operatively. This procedure could have been done using unilateral spinal for the bone graft harvest with interscalene block.

Factors like liver disease can affect the pharmacokinetics of local anaesthetics by diminishing hepatic blood flow or impair the production of liver enzymes needed in amide LA metabolism, this results in elevated levels of amide local anaesthetic compared to patient with normal liver function. However, liver function test was not done in this index case. Hepatitis is unlikely to cause a reduction in hepatic blood flow except when there are features of cirrhosis. The onset time of 1-5minutes in this case report is not in support of hepatitis as contributory because it is expected to cause a delayed onset of more than 5minutes.

Limitation of this study was the inability to assay serum level of local anaesthetic.

Conclusion: Seizure can complicate peripheral nerve block by direct unintentional intravascular deposition. Measures instituted to prevent intravascular deposition are not 100% effective. Therefore vigilant monitoring for the prompt detection of the premonitory signs of local anaesthetic toxicity is desirable.

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